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S5        259   RD S4 (unique items)
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S9         64   AU='SMITH, HILDE' OR AU='SMITH, HILDE E' OR AU='SMITH, HIL-
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S13        41   S12 NOT S7
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0014810140 BIOSIS NO.: 200400190897
Invasion of porcine brain microvascular endothelial cells by ***Streptococcus*** ***suis*** serotype 2.
AUTHOR: Vanier Ghyslaine; Segura Mariela; Friedl Peter; Lacouture Sonia; Gottschalk Marcelo (Reprint)
AUTHOR ADDRESS: GREMIP, Faculte de Medecine Veterinaire, Universite de Montreal, 3200 Rue Sicotte, C.P. 5000, Saint-Hyacinthe, PQ, J2S 7C6, Canada**Canada
AUTHOR E-MAIL ADDRESS: gottschm@medvet.umontreal.ca
JOURNAL: Infection and Immunity 72 (3): p1441-1449 March 2004 2004
MEDIUM: print
ISSN: 0019-9567 (ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: **Streptococcus suis** is an important swine pathogen that mainly causes meningitis and occasionally causes other infections, such as endocarditis, arthritis, and pneumonia. The pathogenesis of ***S*** . ***suis*** infection has not been completely defined. However, in order to cause meningitis, ***S*** . ***suis*** has to cross the blood-brain barrier (BBB) made up of brain microvascular endothelial cells. The objective of this work was to study the interactions of ***S*** . ***suis*** serotype 2 with porcine brain microvascular endothelial cells (PBMEC). The ability of North American and European ***S*** . ***suis*** serotype 2 strains to adhere to PBMEC and, most importantly, to invade PBMEC was demonstrated by using an antibiotic protection assay and was confirmed by electron microscopy. The polysaccharide ***capsule*** of ***S*** . ***suis*** seemed to partially interfere with the adhesion and invasion abilities of the bacterium. Our results showed that intracellular viable ***S*** . ***suis*** could be found in PBMEC up to 7 h after antibiotic treatment. Inhibition studies demonstrated that invasion of PBMEC by ***S*** . ***suis*** required actin microfilaments but not microtubular cytoskeletal elements or active bacterial RNA or protein synthesis. At high bacterial doses, suilysin-positive strains were toxic for PBMEC. The role of suilysin in cytotoxicity was confirmed by using purified suilysin, electron microscopy, and the lack of toxicity of a suilysin-negative mutant. In swine, the invasion of endothelial cells of the BBB could play an important role in the pathogenesis of the meningitis caused by ***S*** . ***suis*** .

0011639781 BIOSIS NO.: 199800434028
Streptococcus suis and group B Streptococcus differ in their interactions with murine macrophages
AUTHOR: Segura Mariela A; Cleroux Patrick; Gottschalk Marcelo (Reprint)
AUTHOR ADDRESS: Groupe Rech. Maladies Infect. Porc, Fac. Med. Vet., Univ. Montreal, PO Box 5000, St-Hyacinthe, PQ J2S 7C6, Canada**Canada
JOURNAL: FEMS Immunology and Medical Microbiology 21 (3): p189-195 July, 1998 **1998**
MEDIUM: print
ISSN: 0928-8244
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: **Streptococcus suis** type 2 and group B Streptococcus type III (GBS) are important encapsulated bacterial species causing meningitis. In the present study we compared quantitatively the uptake and intracellular survival of ***S*** . ***suis*** type 2 and GBS type III with murine macrophages in non-opsonic conditions. The role of the **capsule** of both pathogens was also studied using previously obtained unencapsulated isogenic mutants. Encapsulated ***S*** . **suis** wild-type strain was practically not phagocytosed, while the unencapsulated mutant was easily ingested by macrophages. On the other hand, the well encapsulated GBS strain and its unencapsulated mutant were both phagocytosed in large numbers. Even if ***S*** . ***suis*** unencapsulated mutant showed a higher uptake rate than the parental strain, this value was always markedly lower than the numbers of ingested GBS strains. In addition, the intracellular survival of encapsulated and unencapsulated GBS strains was significantly higher than that of ***S*** . ***suis*** strains. These results suggest that interactions between GBS type III and ***S*** . ***suis*** type 2 with murine macrophages as well as the role of the **capsule** as an antiphagocytic factor are different for the two bacterial pathogens.

0011370845 BIOSIS NO.: 199800165092
Streptococcus suis serotype 2 mutants deficient in **capsular** expression
AUTHOR: Charland Nathalie; Harel Josee; Kobisch Marylene; Lacasse Serge; Gottschalk Marcelo (Reprint)
AUTHOR ADDRESS: Groupe de Recherche sur les Maladies Infectieuses du Porc, Faculte de medecine veterinaire, Universite de Montreal, CP 5000, St-Hyacinthe, PQ J2S 7C6, Canada**Canada
JOURNAL: Microbiology (Reading) 144 (2): p325-332 Feb., 1998 ***1998***
MEDIUM: print
ISSN: 1350-0872
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: **Streptococcus suis** serotype 2 is responsible for a wide variety of porcine infections. In addition, it is considered a zoonotic agent. Knowledge about the virulence factors for this bacterium is limited but its polysaccharide **capsule** is thought to be one of the most important. Transposon mutagenesis with the self-conjugative transposon Tn916 was used to obtain acapsular mutants from the virulent ***S*** . ***suis*** type 2 reference strain S735. Clones were screened by colony-dot ELISA with a monoclonal antibody specific for a type 2 **capsular** epitope and clones that failed to react with the antibody were characterized. Two mutants, 2A and 79, having one and two Tn916 insertions respectively, were chosen for further characterization. Absence of **capsule** was confirmed by coagglutination, capillary precipitation and **capsular** reaction tests and by transmission electron microscopy. Absence of ***capsular*** polysaccharides correlated with increased hydrophobicity and phagocytosis by both murine macrophages and porcine monocytes compared to the wild-type strain. Furthermore, both mutants were shown to be avirulent in murine and pig models of infection. Finally, mutant 2A was readily eliminated from circulation in mice compared to the wild-type strain, which persisted more than 48 h in blood. Thus, isogenic mutants defective in ***capsule*** production demonstrate the importance of **capsular** polysaccharides as a virulence factor for ***S*** . ***suis*** type

0010946926 BIOSIS NO.: 199799580986
Production and characterization of **Streptococcus suis** type 2 mutants deficient in **capsular** expression
AUTHOR: Charland N (Reprint); Harel J (Reprint); Kobisch M; Jacques M (Reprint); Gottschalk M (Reprint)
AUTHOR ADDRESS: Faculty Veterinary Med., Univ. Montreal, St-Hyacinthe, PQ, Canada**Canada
JOURNAL: Abstracts of the General Meeting of the American Society for Microbiology 97 (0): p37 1997 **1997**
CONFERENCE/MEETING: 97th General Meeting of the American Society for Microbiology Miami Beach, Florida, USA May 4-8, 1997; 19970504
ISSN: 1060-2011
DOCUMENT TYPE: Meeting; Meeting Abstract; Meeting Poster
RECORD TYPE: Citation
LANGUAGE: English

0010519529 BIOSIS NO.: 199699153589
Role of **capsular** sialic acid in virulence and resistance to phagocytosis of **Streptococcus suis capsular** type 2
AUTHOR: Charland Nathalie; Kobisch Marylene; Martineau-Doize Beatric; Jacques Mario; Gottschalk Marcelo (Reprint)
AUTHOR ADDRESS: Groupe Rech. Maladies Infect. Porc, Fac. Med. Vet., Univ. Montreal, CP 5000, St. Hyacinthe, PQ J2S 7C6, Canada**Canada
JOURNAL: FEMS Immunology and Medical Microbiology 14 (4): p195-203 1996
1996
ISSN: 0928-8244
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: **Streptococcus suis capsular** type 2 has a ***capsule*** rich in sialic acid (NANA). Sialic acid, known to be an antiphagocytic factor for many bacterial species, inhibits the activation of the alternative complement pathway. The role of ***capsular*** NANA in virulence, resistance to phagocytosis and intracellular survival of ***S*** . ***suis*** ***capsular*** type 2 was evaluated. In general, a low concentration of NANA was observed for all the ***S*** . ***suis*** strains tested. In addition, no difference could be found in NANA concentrations between strains of different virulence degrees. Sialic acid concentration increased in the virulent strain 89-1591 and the avirulent strain 90-1330 after in vivo growth with in increased ***capsular*** material thickness compared to growth in vitro. No significant difference could be found in the phagocytosis rate by porcine blood monocytes of either strain and strain 89-1591 treated with sialidase or the sialic acid-binding, lectin from Sambucus nigra (SNA I). Intracellular survival of strain 89-1591 decreased after treatments with sialidase or lectin. becoming comparable to that of strain 90-1330. Finally, no difference could be seen in virulence using a murine model. even if strain 89-1591 was treated with the enzyme or the lectin. Thus, NANA does not seem to be a critical virulence factor for ***S*** . ***suis*** ***capsular*** type 2.

8212317 Genuine Article#: 258NC Number of References: 21
Title: Hybridization analysis of the gene encoding a hemolysin (suilysin) of **Streptococcus suis** type 2: evidence for the absence of the gene in some isolates
Author(s): Okwumabua O (REPRINT) ; Abdelmagid O; Chengappa MM
Corporate Source: TUSKEGEE UNIV,COLL VET MED NURSING & ALLIED HLTH, DEPT PATHOBIOL/TUSKEGEE//AL/36088 (REPRINT); KANSAS STATE UNIV,COLL VET MED, DEPT PATHOBIOL DIAGNOST MED/MANHATTAN//KS/66506
Journal: FEMS MICROBIOLOGY LETTERS, **1999**, V181, N1 (DEC 1), P113-121
ISSN: 0378-1097 Publication date: 19991201
Publisher: ELSEVIER SCIENCE BV, PO BOX 211, 1000 AE AMSTERDAM, NETHERLANDS
Language: English Document Type: ARTICLE
Abstract: A hemolysin gene was cloned from a virulent strain of ***Streptococcus*** ***suis*** type 2 strain 1933. Analysis of the gene and its product revealed that it is identical to a previously reported hemolysin (suilysin) of ***S*** . ***suis*** type 2. Southern hybridization analysis of the digested total genomic DNA from ***S*** . **suis** with the cloned hemolysin DNA sequences as probe indicated that the hemolysin gene is present as a single copy on the genome. Genomic DNA of 63 isolates of ***S*** . ***suis*** encompassing all known serotypes were examined by DNA hybridization and polymerase chain reaction (PCR) studies for the presence of the hemolysin gene homolog. The results of both techniques were identical and demonstrated the absence of the hemolysin gene in some isolates. In DNA hybridization studies, three DNA probes derived from the hemolysin encoding gene were used. Results showed that sequences encoding the C-terminal 257 amino acid residues (Probe 1) were the most conserved and hybridized to a 1.2 kb fragment in 32 (51%) strains and a 4.0 kb fragment in 23 (36%) strains respectively. Thus, Probe 2 hybridized to the DNA of 55 (87%) of the isolates tested. The first probe (Probe 1) comprising almost the entire hemolysin gene and the third probe (Probe 3) which consisted of the N-terminal sequences hybridized only to a 4.0 kb fragment in 23 (36%) of the strains tested. Eight (13%) of the strains tested were hybridization and PCR negative. The hybridization of the C-terminal end sequences (Probe 2) to the 1.2 kb fragment in 32 (51%) of the strains and the lack of hybridization of the probes to eight (13%) strains may suggest the presence of different types of hemolysin molecule in ***S*** . ***suis*** strains.

0015055074 BIOSIS NO.: 200400425863
Encapsulated **Streptococcus suis** inhibits activation of signaling pathways involved in phagocytosis
AUTHOR: Segura Mariela; Gottschalk Marcelo; Olivier Martin (Reprint)
AUTHOR ADDRESS: Dept Microbiol and Immunol, McGill Univ, Duff Med Bldg,3775 Univ St, Montreal, PQ, H3A 2B4, Canada**Canada
AUTHOR E-MAIL ADDRESS: martin.olivier@staff.mcgill.ca
JOURNAL: Infection and Immunity 72 (9): p5322-5330 September 2004 2004
MEDIUM: print
ISSN: 0019-9567 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: **Streptococcus suis capsular** type 2 is an important zoonotic agent of meningitis. Previous studies reported that, in contrast to nonencapsulated mutants, encapsulated ***S*** . ***suis*** is able to resist phagocytosis. However, the mechanisms by which ***S*** . ***suis*** avoids phagocytosis are unknown. To elucidate the signaling pathway(s) involved in ***S*** . ***suis*** antiphagocytosis, we compared the ability of an encapsulated strain and its nonencapsulated mutant to induce the activation of Akt and protein kinase C (PKC), which are downstream kinases of the phosphatidylinositol 3-kinase (PI-3K) pathway, known to be involved in the phagocytosis processes. The results demonstrated high levels of Akt and PKCalpha phosphorylation after infection of J774 macrophages with the nonencapsulated mutant, whereas the encapsulated strain showed reduced activation of PI-3K/Akt/PKCalpha signaling pathway, as well as several protein tyrosine events. These results correlated with the number of intracellular bacteria. Macrophages pretreated with specific PI-3K or PKC inhibitors showed reduced levels of Akt and PKCalpha phosphorylation, resulting in 50% reduction of phagocytosis. The role of phosphatases in the antiphagocytic mechanisms was evaluated by using phosphatase inhibitors, as well as SHP-1-deficient macrophages. Only in the absence of SHP-1 did the phagocytosis of encapsulated ***S*** . ***suis*** significantly increase, leading to Akt phosphorylation levels similar to those observed with the nonencapsulated strain, indicating activation of this important SH2 domain-containing tyrosine phosphatase by encapsulated ***S*** . ***suis*** . Finally, when purified ***S*** . ***suis*** ***capsular*** polysaccharide (CPS) was added to macrophages, no phosphorylation events were observed. In addition, CPS and encapsulated ***S*** . ***suis*** were able to inhibit the uptake of the nonencapsulated mutant. These results suggest the importance of CPS in the mechanisms, whereby ***S*** . ***suis*** downmodulates phagocytosis.

knock-out (nok′out)

A genetically engineered organism in which the genome has been altered by site-directed recombination so that a gene is deleted.

Prev

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☐	L2	(s adj1 suis) or (streptococcus adj1 suis)	585
☐	L1	streptococcus same suis	465

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